



=> s 16(P) (transfect? or transduc? or transform? or immunogeni?)

8998 TRANSFECT?  
97056 TRANSDUC?  
210927 TRANSFORM?  
7191 IMMUNOGENI?  
L7 5 L6(P) (TRANSFECT? OR TRANSDUC? OR TRANSFORM? OR IMMUNOGENI?)

=> d 17 1-5 date

L7: 1 of 5  
TITLE: MCH4 and MCH5, apoptotic proteases  
US PAT NO: 5,851,815 DATE ISSUED: Dec. 22, 1998  
[IMAGE AVAILABLE]  
APPL-NO: 08/618,408 DATE FILED: Mar. 19, 1996

L7: 2 of 5  
TITLE: Interleukin-2 receptor subunit ectodomain fusion protein  
comprising a leucine zipper domain  
US PAT NO: 5,837,816 DATE ISSUED: Nov. 17, 1998  
[IMAGE AVAILABLE]  
APPL-NO: 08/474,741 DATE FILED: Jun. 7, 1995  
REL-US-DATA: Continuation-in-part of Ser. No. 438,259, May 10, 1995,  
abandoned.

L7: 3 of 5  
TITLE: MCH4 and MCH5, apoptotic protease, nucleic acids encoding  
and methods of use  
US PAT NO: 5,786,173 DATE ISSUED: Jul. 28, 1998  
[IMAGE AVAILABLE]  
APPL-NO: 08/665,220 DATE FILED: Jun. 14, 1996  
REL-US-DATA: Continuation-in-part of Ser. No. 618,408, Mar. 19, 1996.

L7: 4 of 5  
TITLE: Methods of preparing soluble, oligomeric proteins  
US PAT NO: 5,716,805 DATE ISSUED: Feb. 10, 1998  
[IMAGE AVAILABLE]  
APPL-NO: 08/446,922 DATE FILED: May 18, 1995  
REL-US-DATA: Continuation-in-part of Ser. No. 107,353, Aug. 13, 1993,  
abandoned, which is a continuation-in-part of Ser. No.  
969,703, Oct. 23, 1992, abandoned, which is a  
continuation-in-part of Ser. No. 805,723, Dec. 5, 1991,  
abandoned, which is a continuation-in-part of Ser. No.  
783,707, Oct. 25, 1991, abandoned.

L7: 5 of 5  
TITLE: Method of preventing or treating disease characterized by  
neoplastic cells expressing CD40  
US PAT NO: 5,674,492 DATE ISSUED: Oct. 7, 1997  
[IMAGE AVAILABLE]  
APPL-NO: 08/360,923 DATE FILED: Dec. 21, 1994  
REL-US-DATA: Continuation-in-part of Ser. No. 172,664, Dec. 23, 1993,  
abandoned.

=> d 17 1-5 kwic

US PAT NO: 5,851,815 [IMAGE AVAILABLE] L7: 1 of 5

SUMMARY:

BSUM(5)

AVAILABLE]

5. 5,482,692, Jan. 9, 1996, Selective catalytic reduction of nitrogen oxides using a ferrocene impregnated zeolite catalyst; Costandi A. Audeh, et al., 423/239.2 [IMAGE AVAILABLE]

6. 5,451,387, Sep. 19, 1995, Selective catalytic reduction of nitrogen oxides using an iron impregnated zeolite catalyst; Maria D. Farnos, et al., 423/239.2 [IMAGE AVAILABLE]

=> s (accessory or costimulatory) (P) (molecule?) (P) (transduc? or transfect?)

24582 ACCESSORY  
128 COSTIMULATORY  
175940 MOLECULE?  
97056 TRANSDUC?  
8998 TRANSFECT?  
L2 61 (ACCESSORY OR COSTIMULATORY) (P) (MOLECULE?) (P) (TRANSDUC? OR  
TRA NSFECT?)

=> s 12(P) (tumor? or tumour? or cancer)

26041 TUMOR?  
2429 TUMOUR?  
26098 CANCER  
L3 10 L2(P) (TUMOR? OR TUMOUR? OR CANCER)

=> d 13 1-10 date



L3: 1 of 10

TITLE: Tumor cells modified to express B7-2 with increased immunogenicity and uses therefor  
US PAT NO: 5,861,310 DATE ISSUED: Jan. 19, 1999  
[IMAGE AVAILABLE]  
APPL-NO: 08/456,104 DATE FILED: May 30, 1995  
REL-US-DATA: Continuation-in-part of Ser. No. 147,773, Nov. 3, 1993, abandoned.

L3: 2 of 10

TITLE: Tumor cells with increased immunogenicity and uses therefor  
US PAT NO: 5,858,776 DATE ISSUED: Jan. 12, 1999  
[IMAGE AVAILABLE]  
APPL-NO: 08/147,772 DATE FILED: Nov. 3, 1993

L3: 3 of 10

TITLE: Alphavirus vector constructs  
US PAT NO: 5,843,723 DATE ISSUED: Dec. 1, 1998  
[IMAGE AVAILABLE]  
APPL-NO: 08/739,167 DATE FILED: Oct. 30, 1996  
REL-US-DATA: Continuation of Ser. No. 404,796, Mar. 20, 1995, which is a continuation-in-part of Ser. No. 376,184, Jan. 20, 1995, abandoned, which is a continuation-in-part of Ser. No. 348,472, Nov. 30, 1994, abandoned, which is a continuation-in-part of Ser. No. 198,450, Feb. 18, 1994, abandoned, which is a continuation-in-part of Ser. No. 122,791, Sep. 15, 1993, abandoned.

L3: 4 of 10

TITLE: T-cell receptors and their use in therapeutic and diagnostic methods  
US PAT NO: 5,830,755 DATE ISSUED: Nov. 3, 1998  
[IMAGE AVAILABLE]

E5	USPAT	1	KIPRIYANOV, JURY I/IN
E6	USPAT	2	KIPROFF, PETER N/IN
E7	USPAT	1	KIPROSS, STANLEY/IN
E8	USPAT	2	KIPSHAGEN, WALTER/IN
E9	USPAT	1	KIPSHIDZE, NICHOLAS/IN
E10	USPAT	1	KIPSHIDZE, NICHOLAS N/IN
E11	USPAT	2	KIPSHIDZE, NICKOLAI/IN
E12	USPAT	2	KIPSHIDZE, NODAR N/IN

=> e cantwell, mary ?/in

E#	FILE	FREQUENCY	TERM
--	--	-----	-----
E1	USPAT	2	CANTWELL, JOSEPH R/IN
E2	USPAT	1	CANTWELL, LAURENCE W/IN
E3	USPAT	0 -->	CANTWELL, MARY ?/IN
E4	USPAT	1	CANTWELL, NANCY/IN
E5	USPAT	1	CANTWELL, RICHARD F/IN
E6	USPAT	2	CANTWELL, RICHARD F JR/IN
E7	USPAT	9	CANTWELL, ROBERT H/IN
E8	USPAT	15	CANTWELL, ROBERT R/IN
E9	USPAT	2	CANTWELL, ROBERT ROGER/IN
E10	USPAT	1	CANTWELL, ROBERT W/IN
E11	USPAT	1	CANTWELL, THOMAS/IN
E12	USPAT	2	CANTWELL, THOMAS C/IN

=> e sharma, sanjai ?/in

E#	FILE	FREQUENCY	TERM
--	--	-----	-----
E1	USPAT	1	SHARMA, ROSHAN LA/IN
E2	USPAT	1	SHARMA, SAMEER/IN
E3	USPAT	0 -->	SHARMA, SANJAI ?/IN
E4	USPAT	1	SHARMA, SANJAY/IN
E5	USPAT	5	SHARMA, SANJAY B/IN
E6	USPAT	19	SHARMA, SATISH C/IN
E7	USPAT	1	SHARMA, SATISH CHANDER/IN
E8	USPAT	2	SHARMA, SATISH K/IN
E9	USPAT	1	SHARMA, SATYA P/IN
E10	USPAT	1	SHARMA, SATYA PRAKASH/IN
E11	USPAT	4	SHARMA, SHAMLA V/IN
E12	USPAT	1	SHARMA, SHANKAR/IN

=> s e4,e5

1	"SHARMA, SANJAY"/IN
5	"SHARMA, SANJAY B"/IN
L1	6 ("SHARMA, SANJAY"/IN OR "SHARMA, SANJAY B"/IN)

=> d 11 1-6

1. 5,676,912, Oct. 14, 1997, Process for exhaust gas NO<sub>x</sub>, CO, and hydrocarbon removal; **Sanjay B. Sharma**, et al., 423/213.2, 213.5, 213.7 [IMAGE AVAILABLE]

2. 5,589,147, Dec. 31, 1996, Catalytic system for the reduction of nitrogen oxides; Maria D. Farnos, et al., 423/239.2 [IMAGE AVAILABLE]

3. 5,520,895, May 28, 1996, Method for the reduction of nitrogen oxides using iron impregnated zeolites; **Sanjay B. Sharma**, et al., 423/239.2, 239.1 [IMAGE AVAILABLE]

4. 5,517,626, May 14, 1996, Open high speed bus for microcomputer system; Jordan J. Archer, et al., 395/290, 285, 306; 711/118 [IMAGE

3/3/8 (Item 8 from file: 55)  
DIALOG(R)File 55:BIOSIS PREVIEWS(R)  
(c) 1999 BIOSIS. All rts. reserv.

10733653 BIOSIS NO.: 199799354798  
Occurrence of thymic lymphoblastic lymphoma in **CD40 ligand**  
knock-out mice transplanted with bone marrow cells expressing  
retrovirally **transduced CD40 ligand**.

AUTHOR: Brown M P; Zhao J F; Brenner M K  
AUTHOR ADDRESS: Cell Gene Therapy Program, St. Jude Children's Res. Hosp.,  
Memphis, TN, USA

JOURNAL: Blood 88 (10 SUPPL. 1 PART 1-2):p654A 1996

CONFERENCE/MEETING: Thirty-eighth Annual Meeting of the American Society of  
Hematology Orlando, Florida, USA December 6-10, 1996  
ISSN: 0006-4971

3/3/6 (Item 6 from file: 55)  
DIALOG(R)File 55:BIOSIS PREVIEWS(R)  
(c) 1999 BIOSIS. All rts. reserv.

10770784 BIOSIS NO.: 199799391929  
Stimulation of CD40 up-regulates costimulatory molecules on follicular lymphoma (FL) cells and on a CD40 **transduced** multiple myeloma (MM) cell line.

AUTHOR: Buhmann R(a); Michl D(a); Nolte A(a); Emmerich B; Winnacker E-L(a);  
Hallek Michael(a)  
AUTHOR ADDRESS: (a)Genzentrum, Univ. Muenchen, Muenchen, Germany

JOURNAL: Annals of Hematology 73 (SUPPL. 2):pA170 1996

CONFERENCE/MEETING: Annual Congress of the German and the Austrian Society of Hematology and Oncology Duesseldorf, Germany October 3-7, 1996  
ISSN: 0939-5555

RECORD TYPE: Citation

LANGUAGE: English

4/3/12 (Item 4 from file: 154)  
DIALOG(R)File 154:MEDLINE(R)  
(c) format only 1999 Dialog Corporation. All rts. reserv.

08527548 96058917

Study of **CD40 ligand** expression in B-cell chronic lymphocytic leukemia.

Brugnoni D; Rossi G; Tucci A; Cattaneo R; Airo P  
Servizio di Immunologia Clinica, Spedali Civili di Brescia, Italy.  
Haematologica (ITALY) Sep-Oct 1995, 80 (5) p440-2, ISSN  
0390-6078 Journal Code: FYB  
Languages: ENGLISH

s3/7/43, 62, 63, 68, 74

3/7/43 (Item 29 from file: 72)  
DIALOG(R)File 72:EMBASE  
(c) 1999 Elsevier Science B.V. All rts. reserv.

06150151 EMBASE No: 1995181423  
**Tumor necrosis factor ligand superfamily: Involvement in the pathology of malignant lymphomas**  
Gruss H.-J.; Dower S.K.  
DMOAMB, UKRV-RRK, Freie University Berlin, Lindenberger Weg 80, D-13122 Berlin Germany  
Blood ( BLOOD ) (United States) 1995, 85/12 (3378-3404)

CODEN: BLOOA ISSN: 0006-4971  
DOCUMENT TYPE: Journal; Review  
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

The TNF receptor superfamily members are all type I membrane glycoproteins with typical homology in the extracellular domain of variable numbers of cysteine-rich repeats (overall homologies, 25% to 30%). In contrast, the TNF ligand superfamily members (with the exception of LTalpha) are type II membrane glycoproteins with homology to TNF in the extracellular domain (overall homologies, 20%). TNF and LTalpha are trimeric proteins and are composed of beta- strands forming a beta-jellyroll. The homology of the beta-strand regions for the TNF ligand superfamily members suggest a similar beta-sandwich structure and possible trimeric or multimeric complex formation for most or all members. A genetic linkage, as evidence for evolutionary relatedness, is found by chromosomal cluster of TNFR p80, CD30, 4-1BB, and OX40 for 1p36; TNFR p60, TNFR-RP, and CD27 for 12p13; TNF, LTalpha, and LTbeta for 6 (MHC locus); CD27L and 4-1BBL for 19p13; and FASL and OX40L for 1q25. Of the TNF ligand superfamily, TNF, LTalpha, and LTalpha and their receptors (TNFR p60, TNFR p80, and TNFR-RP) interact in a complex fashion of cross-binding. However, the other family members presently have a one ligand/one receptor binding principle (CD27/CD27L, CD30/CD30L, CD40/**CD40L**, 4-1BB/4-1BBL, OX40/gp34, and FAS/FASL). In general, the members of the TNF ligand superfamily mediate interaction between different hematopoietic cells, such as T cell/B cell, T cell/monocyte, and T cell/T cell. Signals can be **transduced** not only through the receptors but also through at least some of the ligands. The **transduced** signals can be stimulatory or inhibitory depending on the target cell or the activation state. Taken together, TNF superfamily ligands show for the immune response an involvement in the induction of cytokine secretion and the upregulation of adhesion molecules, activation antigens, and costimulatory proteins, all known to amplify stimulatory and regulatory signals. On the other hand, differences in the distribution, kinetics of induction, and requirements for induction support a defined role for each of the ligands for T-cell-mediated immune responses. The shedding of members of the TNF receptor superfamily could limit the signals mediated by the corresponding ligands as a functional regulatory mechanism. Induction of cytotoxic cell death, observed for TNF, LTalpha, CD30L, CD95L and 4-1BBL, is another common functional feature of this cytokine family. Further studies have to identify unique versus redundant biologic and physiologic functions for each of the TNF superfamily ligands. Primary H-RS cells can express TNF, LTalpha, and CD27L but not CD30L and **CD40L**, in addition to IL-1alpha, IL-5, IL-6, IL-9, and M-CSF. In addition, H-RS cells express high copy numbers of several cytokine receptors such as IL-2R (p55, p75, and p64

subunits), IL-6R, M-CSFR (c-fms), SCFR (c-kit), CD30, CD40, and TNFRs. Cytokines produced by H-RS cells might support the growth of tufting cells (autocrine growth loop) and/or interact with surrounding reactive bystander cells, particularly T cells. Conversely, H-RS cells might respond to cytokines produced by surrounding reactive normal cells (paracrine growth loop). The different interactions between H-RS cells and surrounding normal, reactive bystander cells, such as lymphocytes, plasma cells, histiocytes, neutrophils, eosinophils, and stromal cells, is characteristic for HD. The expression and biologic effects of a panel of cytokines and their counterpart receptors seem to be involved in the pathobiologic interaction between H-RS cells and particularly lymphocytes, mainly CD4<sup>sup</sup>+ T cells. Detailed analyses have to verify the predicted biologic activities of TNF, LTalpha, CD27L, CD30L, **CD40L**, 4-1BBL, gp34/OX40L, and FASL for the H-RS cell/T-cell interactions with impact on **tumor** growth and pathogenesis of HD. Cytokines and cytokine receptors, including TNF/TNFRs, CD30/CD30L, and CD40/**CD40L**, are clearly critical elements in the pathology of HD and are part of the deregulated network of interactive signals between H-RS cells and surrounding bystander cells with membrane associated and cytokine mediated events. HD is a **tumor** of cytokine-producing cells that is causative for several characteristic clinical and pathologic presentation of HD. The functional role of cytokines for the pathogenesis of NHLs is presently unclear. Malignant NHL cells express, depending on their immunophenotype, several TNF receptor and ligand superfamily members. B-cell NHLs are frequently CD27/CD27L, CD30 or CD30L, CD40, and TNFRs/TNF positive, but T-cell NHLs have expression of CD30, **CD40L**, and TNFRs/TNF.

3/7/62 (Item 17 from file: 154)  
DIALOG(R) File 154: MEDLINE(R)  
(c) format only 1999 Dialog Corporation. All rts. reserv.

09384250 98043307

Antitumor responses induced by transgenic expression of **CD40 ligand**.

Grossmann ME; Brown MP; Brenner MK  
Division of Bone Marrow Transplantation and Cell and Gene Therapy Program, St. Jude Children's Research Hospital, Memphis, TN 38105, USA.  
Hum Gene Ther (UNITED STATES) Nov 1 1997, 8 (16) p1935-43, ISSN 1043-0342 Journal Code: A12

Languages: ENGLISH  
Document type: JOURNAL ARTICLE  
Because **CD40 ligand** (**CD40L**) is a co-stimulator molecule for multiple components of the immune response, we wanted to determine whether transgenic expression of the molecule would increase immune responses against a weakly immunogenic murine **tumor**, neuro-2a. **Tumor** cells were **transduced** with a retroviral construct containing the **CD40L** gene and co-injected with variable numbers of non-**CD40L** **transduced** cells into syngeneic mice. Mice injected with cells that expressed **CD40L** had a significant reduction in average **tumor** size as compared to controls ( $p < 0.0001$ ). In addition, survival of the neuro-2a/**CD40L** mice was 48 days versus 34 days for the neuro-2a/neo controls ( $p < 0.02$ ). Expression of **CD40L** by less than 1.5% of neuro-2a cells was sufficient for significant antitumor effects ( $p < 0.001$ ). These antitumor effects protected mice from subsequent challenge with parental neuro-2a cells. The protective effects of **CD40L** were associated with systemic immunomodulation. In vivo depletion of CD8+ cells abrogated the **CD40L**-mediated antitumor effects. Analysis of spleens from **CD40L**-protected animals showed increased numbers of CD4+ and CD8+ cells, the majority of which co-expressed the activation marker CD25. In addition, an increased number of antigen-presenting cells (APCs) expressed the co-stimulatory molecule CD86. These experiments illustrate that **transducing** even a small percentage of **tumor** cells with **CD40 ligand** can create a long-lasting systemic immune response capable of impeding growth of

unmodified neuroblastoma cells.

3/7/63 (Item 18 from file: 154)  
DIALOG(R) File 154: MEDLINE(R)  
(c) format only 1999 Dialog Corporation. All rts. reserv.

09360546 98077024

Efficient adenovirus-mediated gene **transduction** of normal and leukemic hematopoietic cells.

Huang MR; Olsson M; Kallin A; Pettersson U; Totterman TH  
Department of Clinical Immunology, University Hospital, Uppsala, Sweden.  
Gene Ther (ENGLAND) Oct 1997, 4 (10) p1093-9, ISSN 0969-7128  
Journal Code: CCE

Languages: ENGLISH

Document type: JOURNAL ARTICLE

We evaluated the efficiency of adenovirus-mediated gene transfer into normal and malignant human hematopoietic cells. An E-1 and E-3 deleted, replication-defective recombinant Ad.RSV beta gal vector was used and the **transduction** efficiency was studied at a multiplicity of infection of 13 p.f.u. per cell. Approximately 40-50% of normal monocytes were **transduced**, whereas purified normal resting T cells and B cells were resistant to infection. We showed that 50-80% of primary chronic myeloid leukemia cells (CML, n = 12) were efficiently **transduced** in contrast to CML, successful **transduction** of resting primary chronic B lymphocytic leukemia cells required appropriate preactivation of targeted cells. A novel protocol for the efficient **transduction** of adenovirus into B-CLL cells was presented. We showed that anti-CD40 mAb or **CD40 ligand** acts in synergy with rhIL-4 to enable the **transduction** of approximately 50-75% of B-CLL cells (B-CLL, n = 6). Expression of beta-galactosidase in **transduced** CML cells and B-CLL cells was detected for at least 15 days after **transduction**. The present studies underline the utility of adenovirus vectors for the construction of cytokine gene-modified **tumor** vaccines for the treatment of hematopoietic malignancies such as CML and B-CLL.

3/7/68 (Item 23 from file: 154)  
DIALOG(R) File 154: MEDLINE(R)  
(c) format only 1999 Dialog Corporation. All rts. reserv.

09235112 96313272

Repression of apoptosis in human B-lymphoma cells by **CD40-ligand** and Bcl-2: relationship to the cell-cycle and role of the retinoblastoma protein.

Wang H; Grand RJ; Milner AE; Armitage RJ; Gordon J; Gregory CD  
Department of Immunology, University of Birmingham Medical School, Edgbaston, UK.

Oncogene (ENGLAND) Jul 18 1996, 13 (2) p373-9, ISSN 0950-9232  
Journal Code: ONC

Languages: ENGLISH

Document type: JOURNAL ARTICLE

Using a Burkitt lymphoma cell line to model human B-cell apoptosis in vitro, we observed that crosslinking, by antibody, of cell surface immunoglobulin induced G1 growth-arrest followed by apoptosis. By contrast, cells treated with the Ca(2+)-ionophore, ionomycin, generated apoptotic signals in G2/M as well as in G1. Both ionomycin and anti-immunoglobulin treatment induced rapid dephosphorylation of Rb prior to apoptosis. Apoptosis was repressed following exposure to **CD40-ligand** and was accompanied by hyperphosphorylation of Rb and cell-cycle progression but not Bcl-2 expression. Expression of Bcl-2 protein in stable bcl-2-transfected cells also resulted in repression of apoptosis and anti-immunoglobulin-treated cells no longer underwent growth-arrest. In Bcl-2-expressing cells in which apoptosis was repressed, Rb remained hyperphosphorylated, even during G1-arrest induced by ionomycin. TGF beta

treatment of Bcl-2-expressing cells induced G1-arrest, de-phosphorylation of Rb and apoptosis. These results suggest that the functional activity of Bcl-2 in B-lymphoma cells is dependent upon, or leads to, sustained hyperphosphorylation of Rb and that Rb hyperphosphorylation can be uncoupled from cell-cycle progression.

3/7/74 (Item 29 from file: 154)  
DIALOG(R)File 154:MEDLINE(R)  
(c) format only 1999 Dialog Corporation. All rts. reserv.

09090602 97349051

Protective immunity induced by **tumor** vaccines requires interaction between CD40 and its ligand, CD154.

Mackey MF; Gunn JR; Ting PP; Kikutani H; Dranoff G; Noelle RJ; Barth RJ Jr

Department of Microbiology, Dartmouth Medical School and Norris Cotton Cancer Center, Lebanon, New Hampshire 03756, USA.

Cancer Res (UNITED STATES) Jul 1 1997, 57 (13) p2569-74, ISSN 0008-5472 Journal Code: CNF

Contract/Grant No.: AI26296, AI, NIAID; AI37075, AI, NIAID

Languages: ENGLISH

Document type: JOURNAL ARTICLE

Interactions between CD40 and its ligand, CD154 (**CD40L, gp39**), have been shown to play a central role in the regulation of humoral immunity. Recent evidence suggests that this ligand-receptor pair also plays an important role in the induction of cell-mediated immune responses, including those directed against viral pathogens, intracellular parasites, and alloantigens. The contribution of this ligand-receptor pair to the development of protective immunity against syngeneic **tumors** was evaluated by blocking the *in vivo* function of CD154 or by studying **tumor** resistance in mice genetically deficient in CD40 expression (CD40<sup>-/-</sup>). In the former case, anti-CD154 monoclonal antibody treatment inhibited the generation of protective immune responses after the administration of three potent **tumor** vaccines: irradiated MCA 105, MCA 105 admixed with *Corynebacterium parvum* adjuvant, and irradiated B16 melanoma cells **transduced** with the gene for granulocyte macrophage colony-stimulating factor. Confirmation of the role of CD40/CD154 interactions in **tumor** immunity was provided by the overt **tumor** susceptibility in CD40-deficient mice as compared to that in CD40<sup>+/+</sup> mice. In this case, wild-type but not CD40-deficient mice could be readily protected against live TS/A **tumor** challenge by preimmunization with TS/A admixed with *C. parvum*. These findings suggest a critical role for CD40/CD154 interactions in the induction of cellular immunity by **tumor** vaccines and may have important implications for future approaches to cell-based **cancer** therapies.

Set	Items	Description
S1	216	(CD40L OR CD40(W)LIGAND OR GP39) AND (TRANFECT? OR TRANSFORM? OR TRANSDUC?) AND (TUMOR? OR TUMOUR? OR CANCER)
S2	170	RD S1 (unique items)
S3	107	S1 AND TRANSDUC?
S4	24	S2 AND PY=1995
S5	9	S2 AND PY=1994

3/3/62 (Item 17 from file: 154)  
DIALOG(R) File 154: MEDLINE(R)  
(c) format only 1999 Dialog Corporation. All rts. reserv.

09384250 98043307

Antitumor responses induced by transgenic expression of **CD40**  
**ligand.**

Grossmann ME; Brown MP; Brenner MK  
Division of Bone Marrow Transplantation and Cell and Gene Therapy  
Program, St. Jude Children's Research Hospital, Memphis, TN 38105, USA.  
Hum. Gene Ther. (UNITED STATES) Nov 1 1997, 8 (16) p1935-43, ISSN  
1043-0342 Journal Code: A12  
Languages: ENGLISH

*89*  
*853683*

3/3/43 (Item 29 from file: 72)  
DIALOG(R) File 72:EMBASE  
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06150151 EMBASE No: 1995181423  
**Tumor** necrosis factor ligand superfamily: Involvement in the  
pathology of malignant lymphomas  
Gruss H.-J.; Dower S.K.  
DMOAMB, UKRV-RRK, Freie University Berlin, Lindenberger Weg 80, D-13122  
Berlin Germany  
Blood ( BLOOD ) (United States) 1995, 85/12 (3378-3404)

3/3/33 (Item 19 from file: 72)  
DIALOG(R)File 72:EMBASE  
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06917464 EMBASE No: 1997201909

Protective immunity induced by **tumor** vaccines requires interaction between CD40 and its ligand, CD154

Mackey M.F.; Gunn J.R.; Ting P.P.; Kikutani H.; Dranoff G.; Noelle R.J.; Barth R.J. Jr.

R.J. Barth Jr., Section of General Surgery, Dartmouth-Hitchcock Medical Center, One Medical Center Drive, Lebanon, NH 03756 United States  
Cancer Research ( CANCER RES. ) (United States) 1997, 57/13 (2569-2574)

CODEN: CNREA ISSN: 0008-5472

DOCUMENT TYPE: Journal; Article

LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

3/3/26 (Item 12 from file: 72)  
DIALOG(R)File 72:EMBASE  
(c) 1999 Elsevier Science B.V. All rts. reserv.

07056784 EMBASE No: 1997338628  
Efficient adenovirus-mediated gene **transduction** of normal and  
leukemic hematopoietic cells  
Huang M.R.; Olsson M.; Kallin A.; Pettersson U.; Totterman T.H.  
M.R. Huang, Department of Clinical Immunology, University Hospital, S-751  
85 Uppsala Sweden  
Gene Therapy ( GENE THER. ) (United Kingdom) 1997, 4/10 (1093-1099)

CODEN: GETHE ISSN: 0969-7128  
DOCUMENT TYPE: Journal; Article  
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

3/3/12 (Item 12 from file: 55)  
DIALOG(R) File 55:BIOSIS PREVIEWS(R)  
(c) 1999 BIOSIS. All rts. reserv.

09907345 BIOSIS NO.: 199598362263

**Tumor necrosis factor ligand superfamily:** Involvement in the  
pathology of malignant lymphomas.

AUTHOR: Gruss Hans-Juergen(a); Dower Steven K

AUTHOR ADDRESS: (a)Freie Univ. Berlin, UKRV-RRK, Dep. Med. Oncol. Applied  
Molecular Biol., Lindenberger Weg 80, D-1, Germany

JOURNAL: Blood 85 (12):p3378-3404 1995

ISSN: 0006-4971

DOCUMENT TYPE: Literature Review